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     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 08:59:31 ON 28 MAY 2003
L1           116 S EUKARYOTIC (A)KINASE?
L2            37 S HUMAN AND L1
L3            18 DUP REM L2 (19 DUPLICATES REMOVED)
L4             1 S "EPK-55053"
L5             1 S EPK(A)55053
L6       5960876 S RECOMBINANT OR EXPRESS? OR CLON?
L7            13 S L3 AND L6
L8            13 DUP REM L7 (0 DUPLICATES REMOVED)
                 E CURTIS R A/AU
L9           213 S E3
L10            0 S L1 AND L9
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NEWS 7	Sep 03	JAPIO has been reloaded and enhanced
NEWS 8	Sep 16	Experimental properties added to the REGISTRY file
NEWS 9	Sep 16	CA Section Thesaurus available in CAPLUS and CA
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NEWS 18	Dec 17	Adis Clinical Trials Insight now available on STN
NEWS 19	Jan 29	Simultaneous left and right truncation added to COMPENDEX, ENERGY, INSPEC
NEWS 20	Feb 13	CANCERLIT is no longer being updated
NEWS 21	Feb 24	METADEX enhancements
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NEWS 23	Feb 24	TEMA now available on STN
NEWS 24	Feb 26	NTIS now allows simultaneous left and right truncation
NEWS 25	Feb 26	PCTFULL now contains images
NEWS 26	Mar 04	SDI PACKAGE for monthly delivery of multifile SDI results
NEWS 27	Mar 20	EVENTLINE will be removed from STN
NEWS 28	Mar 24	PATDPAFULL now available on STN
NEWS 29	Mar 24	Additional information for trade-named substances without structures available in REGISTRY
NEWS 30	Apr 11	Display formats in DGENE enhanced
NEWS 31	Apr 14	MEDLINE Reload
NEWS 32	Apr 17	Polymer searching in REGISTRY enhanced
NEWS 33	Apr 21	Indexing from 1947 to 1956 being added to records in CA/CAPLUS
NEWS 34	Apr 21	New current-awareness alert (SDI) frequency in WPIDS/WPINDEX/WPIX
NEWS 35	Apr 28	RDISCLOSURE now available on STN
NEWS 36	May 05	Pharmacokinetic information and systematic chemical names added to PHAR
NEWS 37	May 15	MEDLINE file segment of TOXCENTER reloaded
NEWS 38	May 15	Supporter information for ENCOMPPAT and ENCOMPLIT updated
NEWS 39	May 16	CHEMREACT will be removed from STN
NEWS 40	May 19	Simultaneous left and right truncation added to WSCA
NEWS 41	May 19	RAPRA enhanced with new search field, simultaneous left and

right truncation

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FILE 'LIFESCI' ENTERED AT 08:59:31 ON 28 MAY 2003
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=> s eukaryotic (a)kinase?
L1 116 EUKARYOTIC (A) KINASE?

=> s human and l1
L2 37 HUMAN AND L1

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 18 DUP REM L2 (19 DUPLICATES REMOVED)

=> d 1-18 ibib ab

L3 ANSWER 1 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:397007 HCAPLUS
TITLE: 55053, a novel **human eukaryotic kinase** and uses therefor
INVENTOR(S): Curtis, Rory A. J.
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003042371	A2	20030522	WO 2002-US36967	20021115

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-3690 A 20011115
AB The invention provides isolated nucleic acids molecules, designated EPK-55053 nucleic acid molecules, which encode novel protein kinase polypeptides. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing EPK-55053 nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which an EPK-55053 gene has been introduced or disrupted. The invention still further provides isolated EPK-55053 proteins, fusion proteins, antigenic peptides and anti-EPK-55053 antibodies. Diagnostic, screening, and therapeutic methods utilizing compositions of the invention are also provided.

L3 ANSWER 2 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2003:128287 BIOSIS
DOCUMENT NUMBER: PREV200300128287
TITLE: Functional characterization of Drosophila melanogaster PERK eukaryotic initiation factor 2alpha (eIF2alpha) kinase.
AUTHOR(S): Pomar, Natalia; Berlanga, Juan J.; Campuzano, Sonsoles; Hernandez, Greco; Elias, Monica; de Haro, Cesar (1)
CORPORATE SOURCE: (1) Centro de Biologia Molecular 'Severo Ochoa', Facultad de Ciencias, CSIC-UAM, Cantoblanco, Madrid, 28049, Spain: cdeharo@cbm.uam.es Spain
SOURCE: European Journal of Biochemistry, (January 2003, 2003) Vol. 270, No. 2, pp. 293-306. print.
ISSN: 0014-2956.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Four distinct eukaryotic initiation factor 2alpha (eIF2alpha) kinases

phosphorylate eIF2alpha at S51 and regulate protein synthesis in response to various environmental stresses. These are the hemin-regulated inhibitor (HRI), the interferon-inducible dsRNA-dependent kinase (PKR), the endoplasmic reticulum (ER)-resident kinase (PERK) and the GCN2 protein kinase. Whereas HRI and PKR appear to be restricted to mammalian cells, GCN2 and PERK seem to be widely distributed in eukaryotes. In this study, we have characterized the second eIF2alpha kinase found in Drosophila, a PERK homologue (DPERK). Expression of DPERK is developmentally regulated. During embryogenesis, DPERK expression becomes concentrated in the endodermal cells of the gut and in the germ line precursor cells. Recombinant wild-type DPERK, but not the inactive DPERK-K671R mutant, exhibited an autokinase activity, specifically phosphorylated Drosophila eIF2alpha at S50, and functionally replaced the endogenous Saccharomyces cerevisiae GCN2. The full length protein, when expressed in 293T cells, located in the ER-enriched fraction, and its subcellular localization changed with deletion of different N-terminal fragments. Kinase activity assays with these DPERK deletion mutants suggested that DPERK localization facilitates its in vivo function. Similar to mammalian PERK, DPERK forms oligomers in vivo and DPERK activity appears to be regulated by ER stress. Furthermore, the stable complexes between wild-type DPERK and DPERK-K671R mutant were mediated through the N terminus of the proteins and exhibited an in vitro eIF2alpha kinase activity.

L3 ANSWER 3 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:937303 HCAPLUS
DOCUMENT NUMBER: 138:20443
TITLE: Endocrine disruptor screening using DNA chips of endocrine disruptor-responsive genes
INVENTOR(S): Kondo, Akihiro; Takeda, Takeshi; Mizutani, Shigetoshi; Tsujimoto, Yoshimasa; Takashima, Ryokichi; Enoki, Yuki; Kato, Ikunoshin
PATENT ASSIGNEE(S): Takara Bio Inc., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 386 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2002355079	A2	20021210	JP 2002-69354	20020313

PRIORITY APPLN. INFO.:
JP 2001-73183 A 20010314
JP 2001-74993 A 20010315
JP 2001-102519 A 20010330

AB A method and kit for detecting endocrine-disrupting chems. using DNA microarrays are claimed. The method comprises prepg. a nucleic acid sample contg. mRNAs or cDNAs originating in cells, tissues, or organisms which have been brought into contact with a sample contg. the endocrine disruptor. The nucleic acid sample is hybridized with DNA microarrays having genes affected by the endocrine disruptor or DNA fragments originating in these genes have been fixed. The results obtained are then compared with the results obtained with the control sample to select the gene affected by the endocrine disruptor. Genes whose expression is altered by tri-Bu tin, 4-octaphenol, 4-nonylphenol, di-N-Bu phthalate, dichlorohexyl phthalate, octachlorostyrene, benzophenone, diethylhexyl phthalate, diethylstilbestrol (DES), and 17-.beta. estradiol (E2), were found in mice by DNA chip anal.

L3 ANSWER 4 OF 18 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2002619803 MEDLINE
DOCUMENT NUMBER: 22266135 PubMed ID: 12377121

TITLE: Structure and interactions of PAS kinase N-terminal PAS domain: model for intramolecular kinase regulation.
COMMENT: Comment in: Chem Biol. 2002 Nov;9(11):1165-6
AUTHOR: Amezcua Carlos A; Harper Shannon M; Rutter Jared; Gardner Kevin H
CORPORATE SOURCE: Department of Biochemistry, The University of Texas Southwestern Medical Center, Dallas, TX 75390, USA.
CONTRACT NUMBER: CA-90601 (NCI)
SOURCE: Structure (Camb), (2002 Oct) 10 (10) 1349-61.
Journal code: 101087697. ISSN: 0969-2126.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: PDB-1LL8
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 20021015
Last Updated on STN: 20030418
Entered Medline: 20030417

AB PAS domains are sensory modules in signal-transducing proteins that control responses to various environmental stimuli. To examine how those domains can regulate a **eukaryotic kinase**, we have studied the structure and binding interactions of the N-terminal PAS domain of **human** PAS kinase using solution NMR methods. While this domain adopts a characteristic PAS fold, two regions are unusually flexible in solution. One of these serves as a portal that allows small organic compounds to enter into the core of the domain, while the other binds and inhibits the kinase domain within the same protein. Structural and functional analyses of point mutants demonstrate that the compound and ligand binding regions are linked, suggesting that the PAS domain serves as a ligand-regulated switch for this eukaryotic signaling system.

L3 ANSWER 5 OF 18 MEDLINE
ACCESSION NUMBER: 2001504158 MEDLINE
DOCUMENT NUMBER: 21437970 PubMed ID: 11526204
TITLE: Receptor-like kinases from Arabidopsis form a monophyletic gene family related to animal receptor kinases.
AUTHOR: Shiu S H; Bleecker A B
CORPORATE SOURCE: Department of Botany, University of Wisconsin, Madison, WI 53706, USA.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2001 Sep 11) 98 (19) 10763-8.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200111
ENTRY DATE: Entered STN: 20010913
Last Updated on STN: 20030105
Entered Medline: 20011101

AB Plant receptor-like kinases (RLKs) are proteins with a predicted signal sequence, single transmembrane region, and cytoplasmic kinase domain. Receptor-like kinases belong to a large gene family with at least 610 members that represent nearly 2.5% of Arabidopsis protein coding genes. We have categorized members of this family into subfamilies based on both the identity of the extracellular domains and the phylogenetic relationships between the kinase domains of subfamily members. Surprisingly, this structurally defined group of genes is monophyletic with respect to kinase domains when compared with the other **eukaryotic kinase** families. In an extended analysis, animal receptor kinases, Raf kinases, plant RLKs, and animal receptor

tyrosine kinases form a well supported group sharing a common origin within the superfamily of serine/threonine/tyrosine kinases. Among animal kinase sequences, Drosophila Pelle and related cytoplasmic kinases fall within the plant RLK clade, which we now define as the RLK/Pelle family. A survey of expressed sequence tag records for land plants reveals that mosses, ferns, conifers, and flowering plants have similar percentages of expressed sequence tags representing RLK/Pelle homologs, suggesting that the size of this gene family may have been close to the present-day level before the diversification of land plant lineages. The distribution pattern of four RLK subfamilies on Arabidopsis chromosomes indicates that the expansion of this gene family is partly a consequence of duplication and reshuffling of the Arabidopsis genome and of the generation of tandem repeats.

L3 ANSWER 6 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:268802 BIOSIS
DOCUMENT NUMBER: PREV200000268802
TITLE: Amino acid sequence of putative dsRNA-dependent protein **kinase-eukaryotic** initiation factor 2alpha phosphorylation homology domain of hepatitis C virus and interferon sensitivity.
AUTHOR(S): Watanabe, Hideki (1); Nagayama, Kazuyoshi; Enomoto, Nobuyuki; Kurosaki, Masayuki; Miyasaka, Yuka; Yu, Shin-Han; Sakamoto, Naoya; Ikeda, Takaaki; Izumi, Namiki; Sato, Chifumi
CORPORATE SOURCE: (1) Tokyo Med and Dental Univ, Tokyo Japan
SOURCE: Gastroenterology, (April, 2000) Vol. 118, No. 4 Suppl. 2 Part 1, pp. AASLD A939. print..
Meeting Info.: 101st Annual Meeting of the American Gastroenterological Association and the Digestive Disease Week. San Diego, California, USA May 21-24, 2000 American Gastroenterological Association
. ISSN: 0016-5085.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L3 ANSWER 7 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:26973 BIOSIS
DOCUMENT NUMBER: PREV200000026973
TITLE: Protein synthesis inhibition by flavonoids: Roles of eukaryotic initiation factor 2alpha kinases.
AUTHOR(S): Ito, Takahiko (1); Warnken, Sarah P.; May, W. Stratford
CORPORATE SOURCE: (1) Sealy Center for Oncology and Hematology, Department of Internal Medicine, University of Texas Medical Branch at Galveston, Galveston, TX, 77555-1048 USA
SOURCE: Biochemical and Biophysical Research Communications, (Nov. 19, 1999) Vol. 265, No. 2, pp. 589-594.
ISSN: 0006-291X.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Flavonoids such as genistein and quercetin suppress tumor cell growth in vitro and in vivo. Many metabolic enzymes, including protein kinases, are known to be inhibited by flavonoids, yet the molecular targets and biochemical mechanisms of the tumor growth suppression remain unclear. Here, we find that flavonoids inhibit protein synthesis in both mouse and **human** leukemia cells. This inhibition is associated with phosphorylation of the alpha-subunit of eukaryotic initiation factor 2 (eIF2alpha), a key regulatory mechanism of protein translation. Three mammalian eIF2alpha kinases have been identified: the interferon-inducible double-stranded RNA-dependent kinase (PKR), the heme-regulated inhibitor

(HRI), and the very recently discovered PERK/PEK. We find that all of these eIF2alpha kinases can be activated by quercetin and genistein, indicating redundant roles of the eIF2alpha kinases. Thus, activation of eIF2alpha kinases appears to be a mechanism by which flavonoids can inhibit the growth of tumor and leukemia cells.

L3 ANSWER 8 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:113064 HCAPLUS
DOCUMENT NUMBER: 128:242495
TITLE: Eukaryotic elongation factor 1.delta. is hyperphosphorylated by the protein kinase encoded by the UL13 gene of herpes simplex virus 1
AUTHOR(S): Kaqwaguchi, Yasushi; Van Sant, Charles; Roizman, Bernard
CORPORATE SOURCE: The Marjorie B. Kovler Viral Oncology Laboratories, University of Chicago, Chicago, IL, 60637, USA
SOURCE: Journal of Virology (1998), 72(3), 1731-1736
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The translation elongation factor 1.delta. (EF-1.delta.) consists of two forms, a hypophosphorylated form (apparent Mr, 38,000) and a hyperphosphorylated form (apparent Mr, 40,000). Earlier Y. Kawaguchi et al. (1997) reported that whereas mock-infected cells accumulate the hypophosphorylated form, the hyperphosphorylated form of EF-1.delta. accumulates in cells infected with herpes simplex virus 1. The authors now report that the accumulation of the hyperphosphorylated EF-1.delta. is due to phosphorylation by UL13 protein kinase based on the following observations. (I) The relative amts. of hypo- and hyperphosphorylated EF-1.delta. in Vero cells infected with mutant virus lacking the UL13 gene could not be differentiated from those of mock-infected cells. In contrast, the hyperphosphorylated EF-1.delta. was the predominant form in Vero cells infected with wild-type viruses, a recombinant virus in which the deleted UL13 sequences were restored, or with a virus lacking the US3 gene, which also encodes a protein kinase. (Ii) The absence of the hyperphosphorylated EF-1.delta. in cells infected with the UL13 deletion mutant was not due to failure of post-translational modification of infected-cell protein 22 (ICP22)/US1.5 or of interaction with ICP0, inasmuch as preferential accumulation of hyperphosphorylated EF-1.delta. was obsd. in cells infected with viruses from which the genes encoding ICP22/US1.5 or ICP0 had been deleted. (Iii) Both forms of EF-1.delta. were labeled by 32Pi in vivo, but the prevalence of the hyperphosphorylated EF-1.delta. was dependent on the presence of the UL13 protein. (iv) EF-1.delta. immunopptd. from uninfected Vero cells was phosphorylated by UL13 pptd. by the anti-UL13 antibody from lysates of wild-type virus-infected cells, but not by complexes formed by the interaction of the UL13 antibody with lysates of cells infected with a mutant lacking the UL13 gene. This is the first evidence that a viral protein kinase targets a cellular protein. Together with evidence that ICP0 also interacts with EF-1.delta. reported in the paper cited above, these data indicate that herpes simplex virus 1 has evolved a complex strategy for optimization of infected-cell protein synthesis.

DUPLICATE 2

L3 ANSWER 9 OF 18 MEDLINE
ACCESSION NUMBER: 1998040126 MEDLINE
DOCUMENT NUMBER: 98040126 PubMed ID: 9372844
TITLE: Interaction between DNA-dependent protein kinase and a novel protein, KIP.
AUTHOR: Wu X; Lieber M R
CORPORATE SOURCE: Department of Pathology, Washington University School of Medicine, St. Louis, MO 63110, USA.

SOURCE: MUTATION RESEARCH, (1997 Oct) 385 (1) 13-20.
Journal code: 0400763. ISSN: 0027-5107.

PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199712
ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 20030218
Entered Medline: 19971212

AB DNA-dependent protein kinase (DNA-PKcs) is the only **eukaryotic kinase** activated by DNA ends. Mutation of DNA-PKcs results in murine severe combined immune deficiency in mice and radiation sensitivity. Both the immune and the radiation defects are due to a failure in double-strand break repair. Biochemical studies indicate that DNA-PKcs kinase activity is stimulated by the presence of the DNA end binding protein. Ku. Autophosphorylation of DNA-PKcs results in its inactivation. Based on these studies, DNA-PKcs is presumed to play a direct and important role in the repair of double-strand breaks, but the details of its role are quite unclear. We have done two-hybrid analysis of this entire protein to identify other proteins with which it interacts. Thus far, extensive analysis has only revealed one strong interaction that satisfies both high genetic and biochemical stringency. The interaction is with a novel **human** protein that has 26% amino acid identity with the phosphatase component, calcineurin B. We discuss the interaction of DNA-PKcs with this novel calcium-binding protein family member in the context of possible kinase-phosphatase regulation of DNA end joining.

L3 ANSWER 10 OF 18 MEDLINE
ACCESSION NUMBER: 95333279 MEDLINE
DOCUMENT NUMBER: 95333279 PubMed ID: 7609068
TITLE: Characterization of the novel protein kinase activity present in the R1 subunit of herpes simplex virus ribonucleotide reductase.
AUTHOR: Cooper J; Conner J; Clements J B
CORPORATE SOURCE: MRC Virology Unit, Institute of Virology, Glasgow, United Kingdom.
SOURCE: JOURNAL OF VIROLOGY, (1995 Aug) 69 (8) 4979-85.
Journal code: 0113724. ISSN: 0022-538X.

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199508
ENTRY DATE: Entered STN: 19950828
Last Updated on STN: 20020420
Entered Medline: 19950811

AB We have compared the protein kinase activities of the R1 subunits from herpes simplex virus types 1 (HSV-1) and 2 (HSV-2) ribonucleotide reductase following expression in Escherichia coli. Autophosphorylation activity was observed when kinase assays were performed with immunoprecipitated R1 or proteins purified to homogeneity, and the activity was stimulated by the basic protein protamine. Transphosphorylation of histones or calmodulin by purified or immunoprecipitated HSV-1 and HSV-2 R1 was not observed, and our results suggest that the activities of these two proteins are similar. We further characterized the protein kinase activity of HSV-1 R1 by producing insertion and deletion mutants constructed with a plasmid expressing R1 amino acids 1 to 449. C-terminal deletion analysis identified the catalytic core of the enzyme as comprising residues 1 to 292, and this polypeptide will be useful for structural determinations by X-ray crystallography. Insertion of a 4-amino-acid sequence at sites within the

protein kinase domain identified regions essential for activity; insertions at residues 22 and 112 completely inactivated activity, and an insertion at residue 136 reduced activity sixfold. Similar insertions at residues 257, 262, 292, and 343 had no effect on activity. The ATP analog 5'-fluorosulfonylbenzoyladenosine, which covalently modifies conventional **eukaryotic kinases** at an essential lysine residue within the active site, did label HSV R1, but this labelling occurred outside the N-terminal domain. These data indicate that the HSV R1 kinase is novel and distinct from other eukaryotic protein kinases.

L3 ANSWER 11 OF 18 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 95362695 MEDLINE
DOCUMENT NUMBER: 95362695 PubMed ID: 7635846
TITLE: Phosphorylation of Mycoplasma pneumoniae cytadherence-accessory proteins in cell extracts.
AUTHOR: Krebes K A; Dirksen L B; Krause D C
CORPORATE SOURCE: Department of Microbiology, University of Georgia, Athens 30602, USA.
CONTRACT NUMBER: AI00968 (NIAID)
AI33396 (NIAID)
SOURCE: JOURNAL OF BACTERIOLOGY, (1995 Aug) 177 (15) 4571-4.
Journal code: 2985120R. ISSN: 0021-9193.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199509
ENTRY DATE: Entered STN: 19950921
Last Updated on STN: 19950921
Entered Medline: 19950913

AB A cell-free system was used to characterize the phosphorylation of Mycoplasma pneumoniae proteins HMW1 and HMW2, which are involved in the adherence of this organism to **human** tracheal epithelium during infection. The pH and cation requirements for phosphorylation of HMW1 and HMW2 were determined, and the effects of glycolytic intermediates, cyclic AMP, and **eukaryotic kinase**-phosphatase inhibitors and stimulators on this process were examined. Phosphoamino acid analysis identified serine as the major phosphate acceptor for both HMW1 and HMW2 in this system.

L3 ANSWER 12 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1994:695972 HCAPLUS
DOCUMENT NUMBER: 121:295972
TITLE: Molecular cloning and use of cDNA for HRR25-like eukaryotic protein kinases
INVENTOR(S): Hoekstra, Merl F.
PATENT ASSIGNEE(S): Salk Institute for Biological Studies, USA
SOURCE: PCT Int. Appl., 120 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9417189	A2	19940804	WO 1994-US795	19940121
WO 9417189	A3	19941013		
W: CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2132452	AA	19940804	CA 1994-2132452	19940121
EP 632832	A1	19950111	EP 1994-915331	19940121

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE
JP 07505057 T2 19950608 JP 1994-517227 19940121
PRIORITY APPLN. INFO.: US 1993-8001 A 19930121
WO 1994-US795 W 19940121

AB The cDNAs for **eukaryotic kinases** of casein kinase I class designated as HRR25-like proteins are cloned from Saccharomyces. A method for screening in a DNA library a nucleotide sequence capable of restoring DNA strand break repair using the HRR25-like polypeptides or mutants is disclosed. Also disclosed are methods using the polynucleotides in cell-proliferative disorders.

L3 ANSWER 13 OF 18 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.DUPLICATE 4
ACCESSION NUMBER: 94211930 EMBASE
DOCUMENT NUMBER: 1994211930
TITLE: Protein kinase superfamily - Comparisons of sequence data with three-dimensional structures.
AUTHOR: Wei L.; Hubbard S.R.; Smith R.F.; Ellis L.
CORPORATE SOURCE: Center for Genome Informatics, Inst of Biosciences and Technology, Texas A and M University, 2121 Holcombe,Houston, TX 77030, United States
SOURCE: Current Opinion in Structural Biology, (1994) 4/3 (450-455).
ISSN: 0959-440X CODEN: COSBEF
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The elucidation of the three-dimensional structures of complexes of the catalytic subunit of mouse recombinant cAMP-dependent protein kinase with bound divalent ion, nucleotide and peptide inhibitor provides new insights into the structural organization of the active site of this enzyme and the probable roles of individual residues in catalysis. Further, the structure of a second member of the **eukaryotic kinase** superfamily, **human** cyclin-dependent kinase 2, now provides a first look at both the similarities and the variations in kinase structure.

DUPLICATE 5
L3 ANSWER 14 OF 18 MEDLINE
ACCESSION NUMBER: 94358007 MEDLINE
DOCUMENT NUMBER: 94358007 PubMed ID: 8077302
TITLE: Invasive **human** pituitary tumors express a point-mutated alpha-protein kinase-C.
AUTHOR: Alvaro V; Levy L; Dubray C; Roche A; Peillon F; Querat B; Joubert D
CORPORATE SOURCE: Centre CNRS-INSERM de Pharmacologie et d'Endocrinologie, Montpellier, France.
SOURCE: JOURNAL OF CLINICAL ENDOCRINOLOGY AND METABOLISM, (1993 Nov) 77 (5) 1125-9.
Journal code: 0375362. ISSN: 0021-972X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941013
Last Updated on STN: 19941013
Entered Medline: 19941006

AB Protein kinase-C (PKC) is a ubiquitous **eukaryotic kinase** that plays a key role in transmembrane signaling and influences important cellular processes, such as proliferation. Increases in its activity and expression have been demonstrated in adenomatous **human**

pituitaries, with protein expression being the highest in invasive tumors (1). Moreover, in these same invasive tumors, the mean increase in expression (8.9-fold) does not correlate with the mean increase in activity (2.6-fold), suggesting a dysfunction in PKC in these tumors. Here, we show that the PKC alpha-isoform (alpha PKC) is overexpressed in **human** pituitary tumors. The complete sequencing of the PKC cDNA from four invasive tumors has revealed a point mutation that is absent in the noninvasive tumors analyzed. The point mutation is located at position 294 of the protein, in the V3 region, leading to a substitution of a negatively charged aspartic acid by an apolar glycine. Thus, not only is alpha PKC overexpressed in **human** pituitary tumors, but it is also structurally altered in the invasive subpopulation of these tumors.

L3 ANSWER 15 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1994:269500 BIOSIS
DOCUMENT NUMBER: PREV199497282500
TITLE: PAGE conditions allowing the identification of the residues phosphorylated by HS-CTD kinase.
AUTHOR(S): Trigon, Sylviane (1); Paslaru, Liliana; Morange, M.
CORPORATE SOURCE: (1) Unite de Genetique Moleculaire, Ecole Normale Superieure, 46 rue d'Ulm, 75 230 Paris Cedex 05 France
SOURCE: Revue Roumaine de Biochimie, (1993) Vol. 30, No. 3-4, pp. 147-152.
ISSN: 0001-4214.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Cellular stresses result in a decrease of transcriptional activity and protein synthesis and an increase of heat-shock protein gene expression. These events are preceded by rapid modifications such as an alteration in the pattern of phosphorylated proteins. We have previously shown that a CTD kinase activity is increased after heat-shock treatment (HS-CTD) **kinase**). **Eukaryotic** RNA polymerase II largest subunit contains a C-terminal domain (CTD) formed of SPTSPSY contiguous repeated motifs. HS-CTD kinase activity is detected by in vitro phosphorylation of a synthetic tetramer of the heptapeptide SPTSPSY. We have also determined that only the serines present in the repeated SPTSPSY motif are phosphorylated by the HS-CTD kinase activity. To study which of the three serines are phosphorylated, we have synthesized different peptides, containing one or two SPTSPSY motifs, where serines have been successively replaced by alanines. Using these different peptides, we have been able to show with new PAGE conditions that only the central serine of the motif is phosphorylated. We discuss the way to investigate the role of the amino acids surrounding the phosphorylated residue on the HS-CTD kinase activity.

L3 ANSWER 16 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1992:609979 HCAPLUS
DOCUMENT NUMBER: 117:209979
TITLE: Constitutive expression of **human** double-stranded RNA-activated p68 kinase in murine cells mediates phosphorylation of eukaryotic initiation factor 2 and partial resistance to encephalomyocarditis virus growth
AUTHOR(S): Meurs, Eliane F.; Watanabe, Yoshihiko; Kadereit, Suzanne; Barber, Glen N.; Katze, Michael G.; Chong, Karen; Williams, Bryan R. G.; Hovanessian, Ara G.
CORPORATE SOURCE: Unit Virol. Cell. Immunol., Inst. Pasteur, Paris, 75724, Fr.
SOURCE: Journal of Virology (1992), 66(10), 5805-14
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal

LANGUAGE: English

AB The cDNA encoding interferon-induced **human** double-stranded RNA-activated p68 kinase was expressed in murine NIH 3T3 cells by using the pcDNA1/neo vector. Several stable clones were selected which expressed either the wild-type kinase or an inactive mutant possessing a single amino acid substitution in the invariant lysine 296 in the catalytic domain II. The transfected wild-type kinase showed properties similar to those of the natural kinase, such as subcellular ribosomal localization and dependence on double-stranded RNA for autophosphorylation. Upon infection with encephalomyocarditis virus (EMCV), wild-type- but not mutant-expressing clones were found to partially resist virus growth. Such natural antiviral activity was virus specific, since no inhibition was obsd. in the case of vesicular stomatitis virus infection. In accord with EMCV inhibition, the wild-type p68 kinase was found to be highly phosphorylated during infection. Furthermore, its natural substrate, the small subunit of protein synthesis initiation factor eIF2, was phosphorylated. These results demonstrate that p68 kinase is activated during ECMV infection, leading to reduced virus prodn.

L3 ANSWER 17 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1990:514791 BIOSIS
DOCUMENT NUMBER: BA90:132067
TITLE: ADENOVIRUS INHIBITION OF CELLULAR PROTEIN SYNTHESIS IS PREVENTED BY THE DRUG 2 AMINOPURINE.
AUTHOR(S): HUANG J; SCHNEIDER R J
CORPORATE SOURCE: DEP. BIOCHEM., NEW YORK UNIV. MED. CENTER, NEW YORK, NY 10016.
SOURCE: PROC NATL ACAD SCI U S A, (1990) 87 (18), 7115-7119. CODEN: PNASA6. ISSN: 0027-8424.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB Adenovirus infection results in the suppression of cellular protein synthesis, but the mechanism has not been established. In this report we demonstrate that the shut-off of cellular protein synthesis by adenovirus is prevented in cells by treatment with the drug 2-aminopurine. Treatment with 2-aminopurine is shown to prevent suppression of cellular translation without disrupting the normal viral block in the transport of cellular mRNAs from the nucleus to the cytoplasm. We show that viral suppression of cellular protein synthesis occurs concomitant with activation of the interferon-induced double-stranded RNA-activated inhibitor (DAI), a protein kinase, and phosphorylation of the .alpha. subunit of eukaryotic initiation factor 2 (eIF-2.alpha.), but that prevention of host cell shut-off by 2-aminopurine occurs without a decrease in kinase activity or a dephosphorylation of eIF-2.alpha.. Results are presented that indicate that activation of DAI kinase and phosphorylation of eIF-2.alpha. may be required but are not sufficient to achieve inhibition of cellular protein synthesis during adenovirus infection. We suggest that other events, in particular the modification of additional initiation factors, are likely involved in viral inhibition of cellular translation.

L3 ANSWER 18 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1987:147636 BIOSIS
DOCUMENT NUMBER: BA83:76686
TITLE: A MECHANISM BY WHICH ADENOVIRUS VIRUS-ASSOCIATED RNA-I CONTROLS TRANSLATION IN A TRANSIENT EXPRESSION ASSAY.
AUTHOR(S): AKUSJARVI G; SVENSSON C; NYGARD O
CORPORATE SOURCE: DEP. MED. GENETICS, BIOMED. CENTER, S-751 23 UPPSALA, SWEDEN.
SOURCE: MOL CELL BIOL, (1987) 7 (1), 549-551. CODEN: MCEBD4. ISSN: 0270-7306.
FILE SEGMENT: BA; OLD

LANGUAGE: English

AB The mechanism by which adenovirus virus-associated RNA1 stimulates translational efficiency in a transient-expression assay in 293 cells was investigated. We showed that DNA transfection leads to activation of a protein kinase that phosphorylates the .alpha. subunit of eucaryotic initiation factor 2 and, as a consequence, inhibition of polypeptide chain initiation. Cotransfection of a plasmid encoding adenovirus type 2 virus-associated RNA1 recovered the translational capacity by preventing activation of the kinase.

=> s "EPK-55053"
L4 1 "EPK-55053"

=> d all

L4 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2003 ACS
AN 2003:397007 HCAPLUS
TI 55053, a novel human eukaryotic kinase and uses therefor
IN Curtis, Rory A. J.
PA Millennium Pharmaceuticals, Inc., USA
SO PCT Int. Appl.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C12N
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003042371	A2	20030522	WO 2002-US36967	20021115

PI WO 2003042371 A2 20030522
W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2001-3690 A 20011115

AB The invention provides isolated nucleic acids molecules, designated **EPK-55053** nucleic acid molecules, which encode novel protein kinase polypeptides. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing **EPK-55053** nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which an **EPK-55053** gene has been introduced or disrupted. The invention still further provides isolated **EPK -55053** proteins, fusion proteins, antigenic peptides and anti- **EPK-55053** antibodies. Diagnostic, screening, and therapeutic methods utilizing compositions of the invention are also provided.

=> s EPK(A)55053
L5 1 EPK(A) 55053

=> d ibib ab

L5 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2003:397007 HCAPLUS
TITLE: 55053, a novel human eukaryotic kinase and uses therefor
INVENTOR(S): Curtis, Rory A. J.
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003042371	A2	20030522	WO 2002-US36967	20021115

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-3690 A 20011115

AB The invention provides isolated nucleic acids molecules, designated **EPK-55053** nucleic acid molecules, which encode novel protein kinase polypeptides. The invention also provides antisense nucleic acid molecules, recombinant expression vectors containing **EPK-55053** nucleic acid molecules, host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which an **EPK-55053** gene has been introduced or disrupted. The invention still further provides isolated **EPK -55053** proteins, fusion proteins, antigenic peptides and anti- **EPK-55053** antibodies. Diagnostic, screening, and therapeutic methods utilizing compositions of the invention are also provided.

=> d his

(FILE 'HOME' ENTERED AT 08:59:11 ON 28 MAY 2003)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 08:59:31 ON 28 MAY 2003
L1 116 S EUKARYOTIC (A)KINASE?
L2 37 S HUMAN AND L1
L3 18 DUP REM L2 (19 DUPLICATES REMOVED)
L4 1 S "EPK-55053"
L5 1 S EPK(A)55053

=> s recombinant or express? or clon?
3 FILES SEARCHED...
L6 5960876 RECOMBINANT OR EXPRESS? OR CLON?

=> s l3 and l6
L7 13 L3 AND L6

=> dup rem l7
PROCESSING COMPLETED FOR L7

L8 13 DUP REM L7 (0 DUPLICATES REMOVED)

=> d 1-13 ibib ab

L8 ANSWER 1 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:397007 HCAPLUS
TITLE: 55053, a novel **human eukaryotic kinase** and uses therefor
INVENTOR(S): Curtis, Rory A. J.
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003042371	A2	20030522	WO 2002-US36967	20021115

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-3690 A 20011115
AB The invention provides isolated nucleic acids molecules, designated EPK-55053 nucleic acid molecules, which encode novel protein kinase polypeptides. The invention also provides antisense nucleic acid molecules, **recombinant expression** vectors containing EPK-55053 nucleic acid molecules, host cells into which the **expression** vectors have been introduced, and nonhuman transgenic animals in which an EPK-55053 gene has been introduced or disrupted. The invention still further provides isolated EPK-55053 proteins, fusion proteins, antigenic peptides and anti-EPK-55053 antibodies. Diagnostic, screening, and therapeutic methods utilizing compositions of the invention are also provided.

L8 ANSWER 2 OF 13 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2003:128287 BIOSIS
DOCUMENT NUMBER: PREV200300128287
TITLE: Functional characterization of Drosophila melanogaster PERK eukaryotic initiation factor 2alpha (eIF2alpha) kinase.
AUTHOR(S): Pomar, Natalia; Berlanga, Juan J.; Campuzano, Sonsoles; Hernandez, Greco; Elias, Monica; de Haro, Cesar (1)
CORPORATE SOURCE: (1) Centro de Biologia Molecular 'Severo Ochoa', Facultad de Ciencias, CSIC-UAM, Cantoblanco, Madrid, 28049, Spain: cdeharo@cbm.uam.es Spain
SOURCE: European Journal of Biochemistry, (January 2003, 2003) Vol. 270, No. 2, pp. 293-306. print.
ISSN: 0014-2956.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Four distinct eukaryotic initiation factor 2alpha (eIF2alpha) kinases phosphorylate eIF2alpha at S51 and regulate protein synthesis in response to various environmental stresses. These are the hemin-regulated inhibitor

(HRI), the interferon-inducible dsRNA-dependent kinase (PKR), the endoplasmic reticulum (ER)-resident kinase (PERK) and the GCN2 protein kinase. Whereas HRI and PKR appear to be restricted to mammalian cells, GCN2 and PERK seem to be widely distributed in eukaryotes. In this study, we have characterized the second eIF2alpha kinase found in Drosophila, a PERK homologue (DPERK). **Expression** of DPERK is developmentally regulated. During embryogenesis, DPERK **expression** becomes concentrated in the endodermal cells of the gut and in the germ line precursor cells. **Recombinant** wild-type DPERK, but not the inactive DPERK-K671R mutant, exhibited an autokinase activity, specifically phosphorylated Drosophila eIF2alpha at S50, and functionally replaced the endogenous Saccharomyces cerevisiae GCN2. The full length protein, when **expressed** in 293T cells, located in the ER-enriched fraction, and its subcellular localization changed with deletion of different N-terminal fragments. Kinase activity assays with these DPERK deletion mutants suggested that DPERK localization facilitates its in vivo function. Similar to mammalian PERK, DPERK forms oligomers in vivo and DPERK activity appears to be regulated by ER stress. Furthermore, the stable complexes between wild-type DPERK and DPERK-K671R mutant were mediated through the N terminus of the proteins and exhibited an in vitro eIF2alpha kinase activity.

L8 ANSWER 3 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:937303 HCAPLUS
DOCUMENT NUMBER: 138:20443
TITLE: Endocrine disruptor screening using DNA chips of endocrine disruptor-responsive genes
INVENTOR(S): Kondo, Akihiro; Takeda, Takeshi; Mizutani, Shigetoshi; Tsujimoto, Yoshimasa; Takashima, Ryokichi; Enoki, Yuki; Kato, Ikunoshin
PATENT ASSIGNEE(S): Takara Bio Inc., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 386 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2002355079	A2	20021210	JP 2002-69354	20020313

PRIORITY APPLN. INFO.:

JP 2001-73183	A	20010314
JP 2001-74993	A	20010315
JP 2001-102519	A	20010330

AB A method and kit for detecting endocrine-disrupting chems. using DNA microarrays are claimed. The method comprises prepg. a nucleic acid sample contg. mRNAs or cDNAs originating in cells, tissues, or organisms which have been brought into contact with a sample contg. the endocrine disruptor. The nucleic acid sample is hybridized with DNA microarrays having genes affected by the endocrine disruptor or DNA fragments originating in these genes have been fixed. The results obtained are then compared with the results obtained with the control sample to select the gene affected by the endocrine disruptor. Genes whose **expression** is altered by tri-Bu tin, 4-octaphenol, 4-nonylphenol, di-N-Bu phthalate, dichlorohexyl phthalate, octachlorostyrene, benzophenone, diethylhexyl phthalate, diethylstilbestrol (DES), and 17-.beta. estradiol (E2), were found in mice by DNA chip anal.

L8 ANSWER 4 OF 13 MEDLINE
ACCESSION NUMBER: 2001504158 MEDLINE
DOCUMENT NUMBER: 21437970 PubMed ID: 11526204
TITLE: Receptor-like kinases from Arabidopsis form a monophyletic

gene family related to animal receptor kinases.

AUTHOR: Shiu S H; Bleecker A B
CORPORATE SOURCE: Department of Botany, University of Wisconsin, Madison, WI 53706, USA.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2001 Sep 11) 98 (19) 10763-8. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200111
ENTRY DATE: Entered STN: 20010913
Last Updated on STN: 20030105
Entered Medline: 20011101

AB Plant receptor-like kinases (RLKs) are proteins with a predicted signal sequence, single transmembrane region, and cytoplasmic kinase domain. Receptor-like kinases belong to a large gene family with at least 610 members that represent nearly 2.5% of Arabidopsis protein coding genes. We have categorized members of this family into subfamilies based on both the identity of the extracellular domains and the phylogenetic relationships between the kinase domains of subfamily members. Surprisingly, this structurally defined group of genes is monophyletic with respect to kinase domains when compared with the other **eukaryotic kinase** families. In an extended analysis, animal receptor kinases, Raf kinases, plant RLKs, and animal receptor tyrosine kinases form a well supported group sharing a common origin within the superfamily of serine/threonine/tyrosine kinases. Among animal kinase sequences, Drosophila Pelle and related cytoplasmic kinases fall within the plant RLK clade, which we now define as the RLK/Pelle family. A survey of **expressed** sequence tag records for land plants reveals that mosses, ferns, conifers, and flowering plants have similar percentages of **expressed** sequence tags representing RLK/Pelle homologs, suggesting that the size of this gene family may have been close to the present-day level before the diversification of land plant lineages. The distribution pattern of four RLK subfamilies on Arabidopsis chromosomes indicates that the expansion of this gene family is partly a consequence of duplication and reshuffling of the Arabidopsis genome and of the generation of tandem repeats.

L8 ANSWER 5 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:113064 HCAPLUS
DOCUMENT NUMBER: 128:242495
TITLE: Eukaryotic elongation factor 1.delta. is hyperphosphorylated by the protein kinase encoded by the UL13 gene of herpes simplex virus 1
AUTHOR(S): Kaqwaguchi, Yasushi; Van Sant, Charles; Roizman, Bernard
CORPORATE SOURCE: The Marjorie B. Kovler Viral Oncology Laboratories, University of Chicago, Chicago, IL, 60637, USA
SOURCE: Journal of Virology (1998), 72(3), 1731-1736 CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The translation elongation factor 1.delta. (EF-1.delta.) consists of two forms, a hypophosphorylated form (apparent Mr, 38,000) and a hyperphosphorylated form (apparent Mr, 40,000). Earlier Y. Kawaguchi et al. (1997) reported that whereas mock-infected cells accumulate the hypophosphorylated form, the hyperphosphorylated form of EF-1.delta. accumulates in cells infected with herpes simplex virus 1. The authors now report that the accumulation of the hyperphosphorylated EF-1.delta. is

due to phosphorylation by UL13 protein kinase based on the following observations. (I) The relative amts. of hypo- and hyperphosphorylated EF-1.delta. in Vero cells infected with mutant virus lacking the UL13 gene could not be differentiated from those of mock-infected cells. In contrast, the hyperphosphorylated EF-1.delta. was the predominant form in Vero cells infected with wild-type viruses, a **recombinant** virus in which the deleted UL13 sequences were restored, or with a virus lacking the US3 gene, which also encodes a protein kinase. (Ii) The absence of the hyperphosphorylated EF-1.delta. in cells infected with the UL13 deletion mutant was not due to failure of post-translational modification of infected-cell protein 22 (ICP22)/US1.5 or of interaction with ICP0, inasmuch as preferential accumulation of hyperphosphorylated EF-1.delta. was obsd. in cells infected with viruses from which the genes encoding ICP22/US1.5 or ICP0 had been deleted. (Iii) Both forms of EF-1.delta. were labeled by 32Pi in vivo, but the prevalence of the hyperphosphorylated EF-1.delta. was dependent on the presence of the UL13 protein. (iv) EF-1.delta. immunopptd. from uninfected Vero cells was phosphorylated by UL13 pptd. by the anti-UL13 antibody from lysates of wild-type virus-infected cells, but not by complexes formed by the interaction of the UL13 antibody with lysates of cells infected with a mutant lacking the UL13 gene. This is the first evidence that a viral protein kinase targets a cellular protein. Together with evidence that ICP0 also interacts with EF-1.delta. reported in the paper cited above, these data indicate that herpes simplex virus 1 has evolved a complex strategy for optimization of infected-cell protein synthesis.

L8 ANSWER 6 OF 13 MEDLINE
ACCESSION NUMBER: 1998040126 MEDLINE
DOCUMENT NUMBER: 98040126 PubMed ID: 9372844
TITLE: Interaction between DNA-dependent protein kinase and a novel protein, KIP.
AUTHOR: Wu X; Lieber M R
CORPORATE SOURCE: Department of Pathology, Washington University School of Medicine, St. Louis, MO 63110, USA.
SOURCE: MUTATION RESEARCH, (1997 Oct) 385 (1) 13-20. Journal code: 0400763. ISSN: 0027-5107.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199712
ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 20030218
Entered Medline: 19971212

AB DNA-dependent protein kinase (DNA-PKcs) is the only **eukaryotic kinase** activated by DNA ends. Mutation of DNA-PKcs results in murine severe combined immune deficiency in mice and radiation sensitivity. Both the immune and the radiation defects are due to a failure in double-strand break repair. Biochemical studies indicate that DNA-PKcs kinase activity is stimulated by the presence of the DNA end binding protein. Ku. Autophosphorylation of DNA-PKcs results in its inactivation. Based on these studies, DNA-PKcs is presumed to play a direct and important role in the repair of double-strand breaks, but the details of its role are quite unclear. We have done two-hybrid analysis of this entire protein to identify other proteins with which it interacts. Thus far, extensive analysis has only revealed one strong interaction that satisfies both high genetic and biochemical stringency. The interaction is with a novel **human** protein that has 26% amino acid identity with the phosphatase component, calcineurin B. We discuss the interaction of DNA-PKcs with this novel calcium-binding protein family member in the context of possible kinase-phosphatase regulation of DNA end joining.

L8 ANSWER 7 OF 13 MEDLINE
ACCESSION NUMBER: 95333279 MEDLINE
DOCUMENT NUMBER: 95333279 PubMed ID: 7609068
TITLE: Characterization of the novel protein kinase activity present in the R1 subunit of herpes simplex virus ribonucleotide reductase.

AUTHOR: Cooper J; Conner J; Clements J B
CORPORATE SOURCE: MRC Virology Unit, Institute of Virology, Glasgow, United Kingdom.

SOURCE: JOURNAL OF VIROLOGY, (1995 Aug) 69 (8) 4979-85.
Journal code: 0113724. ISSN: 0022-538X.

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199508
ENTRY DATE: Entered STN: 19950828
Last Updated on STN: 20020420
Entered Medline: 19950811

AB We have compared the protein kinase activities of the R1 subunits from herpes simplex virus types 1 (HSV-1) and 2 (HSV-2) ribonucleotide reductase following **expression** in Escherichia coli. Autophosphorylation activity was observed when kinase assays were performed with immunoprecipitated R1 or proteins purified to homogeneity, and the activity was stimulated by the basic protein protamine. Transphosphorylation of histones or calmodulin by purified or immunoprecipitated HSV-1 and HSV-2 R1 was not observed, and our results suggest that the activities of these two proteins are similar. We further characterized the protein kinase activity of HSV-1 R1 by producing insertion and deletion mutants constructed with a plasmid **expressing** R1 amino acids 1 to 449. C-terminal deletion analysis identified the catalytic core of the enzyme as comprising residues 1 to 292, and this polypeptide will be useful for structural determinations by X-ray crystallography. Insertion of a 4-amino-acid sequence at sites within the protein kinase domain identified regions essential for activity; insertions at residues 22 and 112 completely inactivated activity, and an insertion at residue 136 reduced activity sixfold. Similar insertions at residues 257, 262, 292, and 343 had no effect on activity. The ATP analog 5'-fluorosulfonylbenzoyladenosine, which covalently modifies conventional **eukaryotic kinases** at an essential lysine residue within the active site, did label HSV R1, but this labelling occurred outside the N-terminal domain. These data indicate that the HSV R1 kinase is novel and distinct from other eukaryotic protein kinases.

L8 ANSWER 8 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1994:695972 HCAPLUS
DOCUMENT NUMBER: 121:295972
TITLE: Molecular **cloning** and use of cDNA for HRR25-like eukaryotic protein kinases

INVENTOR(S): Hoekstra, Merl F.
PATENT ASSIGNEE(S): Salk Institute for Biological Studies, USA
SOURCE: PCT Int. Appl., 120 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9417189	A2	19940804	WO 1994-US795	19940121

WO 9417189 A3 19941013
W: CA, JP
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
CA 2132452 AA 19940804 CA 1994-2132452 19940121
EP 632832 A1 19950111 EP 1994-915331 19940121
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE
JP 07505057 T2 19950608 JP 1994-517227 19940121
PRIORITY APPLN. INFO.: US 1993-8001 A 19930121
WO 1994-US795 W 19940121

AB The cDNAs for **eukaryotic kinases** of casein kinase I class designated as HRR25-like proteins are **cloned** from Saccharomyces. A method for screening in a DNA library a nucleotide sequence capable of restoring DNA strand break repair using the HRR25-like polypeptides or mutants is disclosed. Also disclosed are methods using the polynucleotides in cell-proliferative disorders.

L8 ANSWER 9 OF 13 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 94211930 EMBASE
DOCUMENT NUMBER: 1994211930
TITLE: Protein kinase superfamily - Comparisons of sequence data with three-dimensional structures.
AUTHOR: Wei L.; Hubbard S.R.; Smith R.F.; Ellis L.
CORPORATE SOURCE: Center for Genome Informatics, Inst of Biosciences and Technology, Texas A and M University, 2121 Holcombe,Houston, TX 77030, United States
SOURCE: Current Opinion in Structural Biology, (1994) 4/3 (450-455).
ISSN: 0959-440X CODEN: COSBEF
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The elucidation of the three-dimensional structures of complexes of the catalytic subunit of mouse **recombinant** cAMP-dependent protein kinase with bound divalent ion, nucleotide and peptide inhibitor provides new insights into the structural organization of the active site of this enzyme and the probable roles of individual residues in catalysis. Further, the structure of a second member of the **eukaryotic kinase** superfamily, **human** cyclin-dependent kinase 2, now provides a first look at both the similarities and the variations in kinase structure.

L8 ANSWER 10 OF 13 MEDLINE
ACCESSION NUMBER: 94358007 MEDLINE
DOCUMENT NUMBER: 94358007 PubMed ID: 8077302
TITLE: Invasive **human** pituitary tumors **express** a point-mutated alpha-protein kinase-C.
AUTHOR: Alvaro V; Levy L; Dubray C; Roche A; Peillon F; Querat B; Joubert D
CORPORATE SOURCE: Centre CNRS-INSERM de Pharmacologie et d'Endocrinologie, Montpellier, France.
SOURCE: JOURNAL OF CLINICAL ENDOCRINOLOGY AND METABOLISM, (1993 Nov) 77 (5) 1125-9.
Journal code: 0375362. ISSN: 0021-972X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941013
Last Updated on STN: 19941013

Entered Medline: 19941006

AB Protein kinase-C (PKC) is a ubiquitous **eukaryotic kinase** that plays a key role in transmembrane signaling and influences important cellular processes, such as proliferation. Increases in its activity and **expression** have been demonstrated in adenomatous **human** pituitaries, with protein **expression** being the highest in invasive tumors (1). Moreover, in these same invasive tumors, the mean increase in **expression** (8.9-fold) does not correlate with the mean increase in activity (2.6-fold), suggesting a dysfunction in PKC in these tumors. Here, we show that the PKC alpha-isoform (alpha PKC) is overexpressed in **human** pituitary tumors. The complete sequencing of the PKC cDNA from four invasive tumors has revealed a point mutation that is absent in the noninvasive tumors analyzed. The point mutation is located at position 294 of the protein, in the V3 region, leading to a substitution of a negatively charged aspartic acid by an apolar glycine. Thus, not only is alpha PKC overexpressed in **human** pituitary tumors, but it is also structurally altered in the invasive subpopulation of these tumors.

L8 ANSWER 11 OF 13 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1994:269500 BIOSIS
DOCUMENT NUMBER: PREV199497282500
TITLE: PAGE conditions allowing the identification of the residues phosphorylated by HS-CTD kinase.
AUTHOR(S): Trigon, Sylviane (1); Paslaru, Liliana; Morange, M.
CORPORATE SOURCE: (1) Unite de Genetique Moleculaire, Ecole Normale Superieure, 46 rue d'Ulm, 75 230 Paris Cedex 05 France
SOURCE: Revue Roumaine de Biochimie, (1993) Vol. 30, No. 3-4, pp. 147-152.
ISSN: 0001-4214.
DOCUMENT TYPE: Article
LANGUAGE: English

AB Cellular stresses result in a decrease of transcriptional activity and protein synthesis and an increase of heat-shock protein gene **expression**. These events are preceded by rapid modifications such as an alteration in the pattern of phosphorylated proteins. We have previously shown that a CTD kinase activity is increased after heat-shock treatment (HS-CTD) **kinase**). **Eukaryotic** RNA polymerase II largest subunit contains a C-terminal domain (CTD) formed of SPTSPSY contiguous repeated motifs. HS-CTD kinase activity is detected by in vitro phosphorylation of a synthetic tetramer of the heptapeptide SPTSPSY. We have also determined that only the serines present in the repeated SPTSPSY motif are phosphorylated by the HS-CTD kinase activity. To study which of the three serines are phosphorylated, we have synthesized different peptides, containing one or two SPTSPSY motifs, where serines have been successively replaced by alanines. Using these different peptides, we have been able to show with new PAGE conditions that only the central serine of the motif is phosphorylated. We discuss the way to investigate the role of the amino acids surrounding the phosphorylated residue on the HS-CTD kinase activity.

L8 ANSWER 12 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1992:609979 HCAPLUS
DOCUMENT NUMBER: 117:209979
TITLE: Constitutive **expression** of **human** double-stranded RNA-activated p68 kinase in murine cells mediates phosphorylation of eukaryotic initiation factor 2 and partial resistance to encephalomyocarditis virus growth
AUTHOR(S): Meurs, Eliane F.; Watanabe, Yoshihiko; Kadereit, Suzanne; Barber, Glen N.; Katze, Michael G.; Chong, Karen; Williams, Bryan R. G.; Hovanessian, Ara G.

CORPORATE SOURCE: Unit Virol. Cell. Immunol., Inst. Pasteur, Paris, 75724, Fr.

SOURCE: Journal of Virology (1992), 66(10), 5805-14
CODEN: JOVIAM; ISSN: 0022-538X

DOCUMENT TYPE: Journal
LANGUAGE: English

AB The cDNA encoding interferon-induced **human** double-stranded RNA-activated p68 kinase was **expressed** in murine NIH 3T3 cells by using the pcDNA1/neo vector. Several stable **clones** were selected which **expressed** either the wild-type kinase or an inactive mutant possessing a single amino acid substitution in the invariant lysine 296 in the catalytic domain II. The transfected wild-type kinase showed properties similar to those of the natural kinase, such as subcellular ribosomal localization and dependence on double-stranded RNA for autophosphorylation. Upon infection with encephalomyocarditis virus (EMCV), wild-type- but not mutant-**expressing clones** were found to partially resist virus growth. Such natural antiviral activity was virus specific, since no inhibition was obsd. in the case of vesicular stomatitis virus infection. In accord with EMCV inhibition, the wild-type p68 kinase was found to be highly phosphorylated during infection. Furthermore, its natural substrate, the small subunit of protein synthesis initiation factor eIF2, was phosphorylated. These results demonstrate that p68 kinase is activated during ECMV infection, leading to reduced virus prodn.

L8 ANSWER 13 OF 13 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1987:147636 BIOSIS
DOCUMENT NUMBER: BA83:76686
TITLE: A MECHANISM BY WHICH ADENOVIRUS VIRUS-ASSOCIATED RNA-I CONTROLS TRANSLATION IN A TRANSIENT **EXPRESSION** ASSAY.

AUTHOR(S): AKUSJARVI G; SVENSSON C; NYGARD O
CORPORATE SOURCE: DEP. MED. GENETICS, BIOMED. CENTER, S-751 23 UPPSALA, SWEDEN.

SOURCE: MOL CELL BIOL, (1987) 7 (1), 549-551.
CODEN: MCEBD4. ISSN: 0270-7306.

FILE SEGMENT: BA; OLD
LANGUAGE: English

AB The mechanism by which adenovirus virus-associated RNA1 stimulates translational efficiency in a transient-**expression** assay in 293 cells was investigated. We showed that DNA transfection leads to activation of a protein kinase that phosphorylates the .alpha. subunit of eucaryotic initiation factor 2 and, as a consequence, inhibition of polypeptide chain initiation. Cotransfection of a plasmid encoding adenovirus type 2 virus-associated RNA1 recovered the translational capacity by preventing activation of the kinase.

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FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 08:59:31 ON 28 MAY 2003

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L2	37	S HUMAN AND L1
L3	18	DUP REM L2 (19 DUPLICATES REMOVED)
L4	1	S "EPK-55053"
L5	1	S EPK(A)55053
L6	5960876	S RECOMBINANT OR EXPRESS? OR CLON?
L7	13	S L3 AND L6
L8	13	DUP REM L7 (0 DUPLICATES REMOVED)

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E1          615      CURTIS R/AU
E2            1      CURTIS R 3RD/AU
E3          213 --> CURTIS R A/AU
E4           78      CURTIS R A J/AU
E5           14      CURTIS R B/AU
E6           19      CURTIS R C/AU
E7            3      CURTIS R C H/AU
E8            2      CURTIS R CAROLYN/AU
E9           30      CURTIS R D/AU
E10         251      CURTIS R E/AU
E11           4      CURTIS R EUGENE/AU
E12         127      CURTIS R F/AU

=> s e3
L9          213 "CURTIS R A"/AU

=> s l1 and l9
L10           0 L1 AND L9

=> d his

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     LIFESCI' ENTERED AT 08:59:31 ON 28 MAY 2003
L1          116 S EUKARYOTIC (A)KINASE?
L2           37 S HUMAN AND L1
L3           18 DUP REM L2 (19 DUPLICATES REMOVED)
L4            1 S "EPK-55053"
L5            1 S EPK(A)55053
L6      5960876 S RECOMBINANT OR EXPRESS? OR CLON?
L7           13 S L3 AND L6
L8           13 DUP REM L7 (0 DUPLICATES REMOVED)
                E CURTIS R A/AU
L9          213 S E3
L10           0 S L1 AND L9
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	Issue Date	Pages	Document ID	Title
1	20020924	5	US 6455270 B1	Method for inhibiting eukaryotic protein kinases
2	20001017	5	US 6132984 A	Method for inhibiting eukaryotic protein kinases

	Issue Date	Pages	Document ID	Title
1	20020625	14	US 6410706 B1	Nucleic acid encoding chitin-binding receptor kinase

	Issue Date	Pages	Document ID	Title
1	20030522	332	US 20030096305 A1	Novel human membrane-associated protein and cell surface protein family members
2	20030515	68	US 20030092048 A1	84604 and 84614, human anion transporter family members and uses therefor
3	20030501	66	US 20030082718 A1	52908, a human potassium channel, and uses thereof
4	20030424	49	US 20030077748 A1	96829, a human transporter family member and uses therefor
5	20030417	59	US 20030073658 A1	47619 and 47621, human ion channels, and uses thereof
6	20030320	77	US 20030054453 A1	68723, sodium/glucose cotransporter family members and uses therefor
7	20030320	47	US 20030054449 A1	63744, a human sugar transporter family member and uses thereof
8	20030313	66	US 20030050441 A1	49938, a novel human phospholipid transporter and uses therefor
9	20030313	58	US 20030049727 A1	25658, a novel human calcium channel subunit and uses thereof
10	20030313	64	US 20030049724 A1	52906, 33408, and 12189, novel potassium channel family members and uses thereof

	Issue Date	Pages	Document ID	Title
11	20030313	51	US 20030049664 A1	87144, human amino acid transporter family member and uses therefor
12	20030306	53	US 20030044933 A1	96895, a human sodium-hydrogen exchanger family member and uses therefor
13	20030227	55	US 20030039991 A1	46798, a human matrix metalloproteinase and uses therefor
14	20030213	98	US 20030032091 A1	48120, 23479 and 46689, novel human hydrolases and uses thereof
15	20030213	52	US 20030032021 A1	44589, a novel human ABC transporter family member and uses thereof
16	20030130	118	US 20030022286 A1	Novel transporter-like genes and uses therefor
17	20030130	51	US 20030022219 A1	85080, a human metal ion transporter family member and uses thereof
18	20030130		US 20030022212 A1	65649, a human metalloprotease family member and uses therefor
19	20030130		US 20030022205 A1	98359, a sodium channel beta 4 subunit, and uses therefor

	Issue Date	Pages	Document ID	Title
20	20030130		US 20030022195 A1	59914 and 59921, choline transporters and uses therefor
21	20030109		US 20030009024 A1	46584, a human transporter family member and uses therefor
22	20030102		US 20030003539 A1	67108, a human phospholipid transporter family member and uses therefor
23	20021219		US 20020193582 A1	69624, a novel human transporter family member and uses therefor
24	20021212		US 20020187524 A1	8099, 46455, 54414, 53763, 67076, 67102, 44181, 67084FL, and 67084 alt, human proteins and methods of use thereof
25	20021205		US 20020182636 A1	53010, a novel human carboxylesterase family member and uses thereof
26	20021128		US 20020177148 A1	FBH58295FL, a novel human amino acid transporter and uses thereof
27	20021121		US 20020173455 A1	23927, a novel human ion channel
28	20021114		US 20020168713 A1	46980, a novel human neuroligin family member and uses thereof
29	20021114		US 20020168668 A1	14691, a human glutamate receptor family member and uses therefor
30	20021107		US 20020165357 A1	38554, 57301 and 58324, human organic ion transporters and uses therefor

	Issue Date	Pages	Document ID	Title
31	20021107		US 20020164769 A1	32144, a novel human fatty acid amide hydrolase family member and uses thereof
32	20021031		US 20020160453 A1	Novel gene encoding a sodium channel beta-3 subunit protein
33	20021024	83	US 20020156253 A1	48000 and 52920, novel human calcium channels and uses thereof
34	20021024		US 20020156002 A1	32620, a novel human sodium-sugar symporter family member and uses thereof
35	20021017		US 20020150978 A1	46798, a novel human matrix metalloproteinase and uses therefor
36	20021017		US 20020150910 A1	33410, a novel human carboxylesterase family member and uses thereof
37	20021010		US 20020146800 A1	48921, a novel human GTP releasing factor and uses therefor
38	20020919		US 20020132785 A1	13305 novel protein kinase molecules and uses therefor
39	20020919		US 20020132303 A1	69318, a human sodium/calcium exchanger (transporter) family member and uses therefor

	Issue Date	Pages	Document ID	Title
40	20020919		US 20020132301 A1	25466, a human transporter family member and uses therefor
41	20020919	111	US 20020132298 A1	67118, 67067, and 62092, human proteins and methods of use thereof
42	20020912		US 20020127650 A1	32468, a human sugar transporter family member and uses therefor
43	20020905		US 20020123098 A1	55063, a novel human NMDA family member and uses thereof
44	20020905		US 20020123097 A1	63760, a novel human transporter and uses thereof
45	20020905		US 20020123094 A1	57250, a novel human sugar transporter family member and uses thereof
46	20020829	71	US 20020119555 A1	53014, a human metalloprotease family member and uses therefor
47	20020829	76	US 20020119547 A1	58569 and 50111, human proteins and methods of use thereof
48	20020829		US 20020119523 A1	67073, a human phospholipid transporter family member and uses therefor
49	20020808		US 20020107373 A1	49937, 49931, and 49933, novel human transporter family members and uses thereof
50	20020808		US 20020107192 A1	23686, a novel human aminotransferase and uses therefor

	Issue Date	Pages	Document ID	Title
51	20020801		US 20020103351 A1	32146 and 57259, novel human transporters and uses therefor
52	20020725		US 20020099197 A1	NOVEL POTASSIUM CHANNEL MOLECULES AND USES THEREFOR
53	20020711		US 20020091238 A1	54370, a novel human sulfate transporter and uses therefor
54	20020711		US 20020090710 A1	57800, a novel human cadherin and uses thereof
55	20020627		US 20020082210 A1	56201, a novel human sodium ion channel family member and uses thereof
56	20020627		US 20020081658 A1	18610, a novel human transient receptor and uses thereof
57	20020627		US 20020081657 A1	21784, a novel human calcium channel family member and uses thereof
58	20020627		US 20020081610 A1	Assays and materials for embryonic gene expression
59	20020627		US 20020081599 A1	57809 and 57798, novel human cadherin molecules and uses therefor
60	20020620		US 20020077462 A1	33556, a novel human transporter and uses thereof
61	20020620		US 20020077312 A1	3700, a novel human protein kinase and uses therefor
62	20020620		US 20020076786 A1	25869, a novel human carboxylesterase and uses thereof
63	20020620		US 20020076757 A1	57805, a novel human cadherin family member and uses thereof

	Issue Date	Pages	Document ID	Title
64	20020523		US 20020062015 A1	54372, a novel human anion transporter and uses therefor
65	20020411	84	US 20020042099 A1	2504, 15977, and 14760, novel protein kinase family members and uses therefor
66	20020321		US 20020035056 A1	54420, a novel human calcium channel
67	20020321		US 20020034801 A1	22105, a novel human thioredoxin family member and uses thereof
68	20020307		US 20020028494 A1	57256 and 58289, novel human transporters and uses thereof
69	20030211		US 6518398 B1	ERG potassium channel
70	20020702		US 6413757 B1	25312, a novel human agmatinase-like homolog
71	20020604		US 6399349 B1	Human aminopeptidase P gene
72	20000822		US 6106826 A	Replication competent, avirulent Herpes simplex virus as a vector for neural and ocular gene therapy
73	19991109		US 5981299 A	Mammalian pancreatic cholesterol esterase
74	19990921		US 5955330 A	Means for enhancing gene expression

	Issue Date	Pages	Document ID	Title
75	19990126	46	US 5863532 A	Compositions and methods comprising cytostatic protein kinase
76	19980811		US 5792832 A	Peptides from mammalian pancreatic cholesterol esterase
77	19970708		US 5646251 A	Alloreaction-associated antigen (ARAG): a novel member of the immunoglobulin gene superfamily
78	19970429		US 5624836 A	DNA encoding bovine pancreatic cholesterol esterase
79	19970218		US 5604118 A	Eukaryotic expression vector system
80	19961231		US 5589456 A	Granulocyte-colony stimulating factor receptors
81	19950606		US 5422248 A	DNA sequences encoding granulocyte-colony stimulating factor receptors
82	19921222		US 5173408 A	Mammalian pancreatic cholesterol esterase
83	19920428		US 5108910 A	DNA sequences encoding fusion proteins comprising GM-CSF and IL-3

	Issue Date	Pages	Document ID	Title
84	19911217		US 5073627 A	Fusion proteins comprising GM-CSF and IL-3

	L #	Hits	Search Text
1	L1	0	"epk-55053"
2	L2	0	epk adj "55053"
3	L3	8	eukaryotic adj kinase$2
4	L4	341034	human
5	L5	2	l3 same l4
6	L6	526704	clon$3 or recombinant or express$3
7	L7	1	l3 same l6
8	L8	5005	curtis.in.
9	L9	0	l3 and l8
10	L10	34525	kinase$3
11	L11	105	l8 and l10
12	L12	84	l11 and eukaryotic

	L #	Hits	Search Text
13	L13	29	"55053"
14	L14	0	l12 and l13